The glue of (ab)normal mental life: Networks of interacting thoughts, feelings and behaviors
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Chapter 9

Discussion

The reality of psychological constructs such as mental disorders and normal personality traits is not adequately captured by common cause models and their psychometric equivalent, latent variable models. In this dissertation, I have outlined a novel perspective, the network perspective, in which thoughts, feelings and behaviors directly interact with one another. In the case of mental disorders, the network approach is promising in that has the potential to explain many empirical phenomena such as comorbidity, the relation between stressful life events and individual symptoms of major depression, and spontaneous recovery. In the case of normal personality traits, the network approach offers an alternative theory of traits, in which they are a consequence of direct interactions between thoughts, feelings and behaviors. Additionally, the network perspective on normal personality has shown promise in linking genes to specific personality components as well as offering an alternative conceptualization of the relation between normal personality and psychopathology. While introducing the network perspective and outlining its viability for mental disorders and normal personality was the central aim of this dissertation, it is now time to take a step back and consider (1) (other) psychological constructs for which a network perspective might (not) be accurate and (2) some methodological obstacles that need to be overcome in order for firmly establishing network models as part of the psychometric toolbox.

Networks: Yes or no?

Future work will need to elucidate for exactly which disorders the network perspective is the best explanation and for which it is not. Throughout this dissertation, major depression was the prime example of a disorder for which we think a network model might be accurate in explaining its pathogenesis. Other examples include panic disorder. Panic disorder has four symptoms (APA, 1994): (1) recurrent unexpected panic attacks; (2) at least one of the attacks has been followed by one month (or more) of one (or more) of (2a) persistent concern about having additional attacks, or (2b) worry about the implications of the attack or its consequences (e.g., losing control, having a heart attack, “going crazy”); and (3) there is a significant change in behavior related to the attacks (e.g., avoiding public places: agoraphobia). Here, as explicated in Borsboom

(2008b), a possible causal chain leading up to the pathogenesis of panic disorder is easily envisioned: $1 \to 2a \to 3$ and $1 \to 2b \to 3$. Another example of a disorder for which the network model might paint a realistic picture of its pathogenesis is bipolar disorder, especially the clinical manifestations in which patients switch between (hypo)mania and (sub-threshold) depression. For example, empirical research suggests that sleep disturbances are intimately associated with both the onset of a manic phase (i.e., decreased need for sleep is regularly observed before the mood switch to mania: e.g., Bauer et al., 2006; Leibenluft, Albert, Rosenthal, & Wehr, 1996; Plante & Winkelman, 2008) as well as with reducing depressive symptoms (i.e., sleep deprivation has antidepressant effects: e.g., Bunney & Bunney, 2012; J. Gillin, 1983; Hemmeter, Hemmeter-Spernal, & Krieg, 2010; Kuhs & Tölle, 1991; Landsness, Goldstein, Peterson, Tononi, & Benca, 2011; Wirz-Justice & van den Hoofdakker, 1999; Wu & Bunney, 1990). That is, a symptom (i.e. sleep disturbances) might be an important bridge between manic and depressive states, an observation that sits well with network models, as was shown in Chapter 2.

The origins of other disorders might not be predominantly founded upon symptom-symptom interactions. For example, in the case of development of affective symptoms after traumatic brain injury, it might be so that the symptoms are caused by the brain injury. That is, especially when the traumatic brain injury is located in the left anterior region, the injury itself might be the common cause of affective symptoms, which might culminate into an episode of major depression (Ownsworth & Oei, 1998). This is notably different from the situation in which affective symptoms are developed because of an inability to cope with the consequences of the traumatic brain injury (e.g., a person cannot adjust to the fact that the injury has resulted in the inability to return to work). In that case, the traumatic brain injury is a stressful life event for which we have evidence (see Chapter 4) that they can influence individual affective symptoms. In other disorders, it might be the case that symptom-symptom interactions are crucial in maintaining a pathological condition such as addiction (e.g., substance use $\to$ being broke $\to$ stealing from sister to buy substance $\to$ legal problems $\to$ substance use) but that the initiation of repeated substance use has its roots in an imbalance between dopaminergic circuits that underlie reward and conditioning and those that underlie executive functioning (Volkow, Wang, Fowler, Tomasi, & Telang, 2011).

Mental disorders, as we have portrayed them in our network models, are instances where an otherwise normal functioning system shows pathological behavior. That is, the nodes themselves are not pathological—not sleeping and feeling blue from time to time is normal and happens to virtually everyone—but for some ranges of parameter values (e.g., strong connections between symptoms) the behavior of the system becomes pathological. This is in line with how Mackey and Glass (1977) define a ‘dynamical disease’: “...characterized by the operation of a basically normal control system in a region of physiological parameters that produces pathological behavior”. There are, however, probably disorders for which this definition of a disorder is inaccurate. Take, for example, psychopathy (e.g. Hare, 2003): psychopathy is a personality disorder that is characterized by symptoms such as pathological lying, manipulative, grandiose sense of self-worth and superficial charm. Now, for these types of symptoms, it is hard to argue that the symptoms are themselves normal and that it is the behavior of the system that is pathological in the case of psychopaths. To the contrary, in this case, it appears to make more sense to say that the symptoms themselves are pathological: most people are not manipulative and do not have a grandiose sense of self-worth. Another example is psychosis: a person who suffers from psychosis might have hallucinations (e.g., hearing the voice of your dead grandmother) or delusions (e.g., being convinced that your apartment is bugged by the CIA), both of which are symptoms that, as in the case of psychopathy, are themselves not normal: most healthy people will not have ‘hallucinations’ or ‘delusions’
in their psychopathology networks (although the prevalence for hearing voices, for example, is higher—around 10%—than the prevalence of schizophrenia—around 1% van Os, Hanssen, Bijl, & Vollebergh, 2001). For these and similar disorders, the way we conceptualize and mathematically formalize corresponding network models might be inaccurate. Because the symptoms themselves are pathological, a network model for these disorders should, for instance, incorporate nodes that are continuous. For such nodes, we would subsequently have to show that, for example, simulation models in which the connection weights are equal across networks, activated nodes in the extreme range of the continuum (i.e., in the pathological range) are capable of producing pathological behavior of the system as a whole. On the other hand, one might question whether a network perspective, even if the weights are assumed to be equal for everyone, makes sense in the first place: if psychosis, psychopathy and similar disorders are pathological just because their respective symptoms are pathological and as a result, make it hard to function normally in our society, regardless of whether these symptoms interact with one another; then why the interactions? And without interactions, there is no network.

Methodological issues

One key issue for network theory to advance is the validation of techniques that are now in use to construct and analyze networks. We need to make sure, for example, that constructing networks in which the edges represent partial correlations and regression weights, are unaffected by, for example sample size and variance of the nodes. Getting this right is particularly important for future research endeavors aimed at elucidating differences in architecture between healthy people and people burdened with a particular mental disorder: what makes networks vulnerable to end up in a pathological state? When, for example, comparing the network of a healthy group with the network of a patient group (and the sample sizes of these groups are unequal), it is important to verify that differences between these networks are due to true differences in architecture instead of being the result of differences in architecture of the result of differences in, say, sample size: when defining an edge to be drawn whenever a (partial) correlation is significant, the network based on the larger sample size will contain more edges than the network based on the smaller sample size; but not necessarily because the architecture of these networks truly differs but because the larger the sample size, the more correlations are significant. Considering variance of the nodes, when for example comparing networks at multiple time points in which the edges are based on correlations, one could observe that correlations are increasing over time. This might be an indication that the connections between symptoms are truly becoming stronger. However, alternatively, it might also be an indication of restriction of range: when the variance of nodes is decreasing over time, this results in stronger correlations over time, which, in the case of restriction of range, have nothing to do with a true increase of connectivity between nodes.

Additionally, the adequacy of other techniques for determining the connectivity of a network should be investigated. For example, a common way of determining whether or not a causal relation exists between variables in econometrics is (non)linear Granger causality (Diks & Panchenko, 2005, 2006; Granger, 1969; Hiemstra & Jones, 1994). When considering a strictly stationary and weakly dependent bivariate time series process \( \{(X_t, Y_t)\}, t \in \mathbb{Z}\), \(\{X_t\} \) is said to Granger cause \(\{Y_t\} \) if \(F(Y_t|I_{t-1}) \neq F(Y_t|I_{t-1} - X_{t-1})\) where \(F\) denotes a conditional probability distribution and \(I_{t-1}\) denotes a vector containing past values of \(X\) and \(Y\) (length of vector \(I\) depends on the length of the lag). Thus, Granger causality denotes the situation where the conditional probability distribution of \(Y_{t-1}\) given past values of \(X\) and \(Y\) is not equivalent to the conditional probability distribution of \(Y_{t-1}\) given only past values of \(Y\): past values of \(X\) contain additional
information about current and future values of Y. In the linear multivariate Gaussian case, Granger causality can be relatively easily determined based on the significance of the coefficients of, for example, a vector autoregressive model. As such, when assuming linearity in the case that one has intensive time series data, one can use this technique to determine which nodes in a network might be causally related. In the non-linear (i.e., non-parametric) case, it is, at present, not possible to determine Granger causality for more than two time series.

Another important issue is the correspondence between inter- and intra-individual networks. While I have emphasized throughout the dissertation that collecting time-intensive data is the key to elucidating pathological mechanisms in individuals, the reality is that we still often deal with cross-sectional, inter-individual data. When estimating network parameters for such inter-individual data, is there anything one can conclude about the collection of intra-individual networks that underly the between-subjects network? For example, one might find in a large sample that the connection between depressed mood and thoughts of suicide is particularly strong. Does this finding reveal anything about the networks of the individuals that make up the sample: does it mean, for example, that, on average, most individuals in the sample also have a strong connection between depressed mood and thoughts of suicide in their network? It is probably too simple to, for instance, assume that individuals, in the binary case, who have a “1” for both symptoms necessarily have a strong connection between these symptoms. In future efforts, we will need to search for models with which we can estimate the likelihood of a response pattern (e.g., “1” for both depressed mood and thoughts of suicide) given a certain between-subjects network architecture. As such, we would be able to determine what intra-individual response patterns correspond to between-subjects connectivity (e.g., a “0” “1” response pattern is most likely for the connection between insomnia and fatigue with regression coefficient 1.2) and accordingly.

A final key issue concerns developing ways of quantifying the similarity of networks. For example, when one has constructed between-subjects networks for various time points during a clinical trial with an antidepressant, it might be interesting to determine whether the drug promotes actual change in the networks’ architecture. When the connections are unweighted, the Jaccard index might be a likely candidate (Levandowsky & Winter, 1971), which is defined as the intersection of two sets, A and B, divided by their union: $J = \frac{(A \cap B)}{(A \cup B)}$. So for example, when both network A and B have 10 edges in total and they have five of them in common, $J = 5/15=1/3$. For weighted connections, $J$ is not suited since, for example, $J$ would be one if two networks are completely similar in terms of which connections are present, but this could obscure the fact that in network A, these connections are much stronger. A solution might be to weigh the union and intersection with the adjacency matrices of both networks (Gamallo, Gasperin, Agustini, & Lopes, 2001). In the case of two (or more) different and independent samples, for example when one wants to know whether the major depression architecture differs for men and women, other options exist. For example, one might construct a multi-group analysis in which the relative fit of a model in which network parameters (e.g., regression weights) are constrained to be equal across samples is compared to a model in which these parameters are estimated separately in both samples.

A final note

Humans are complex as are their thoughts, feelings and behaviors. In order to come to understand a small part of this complexity, it is necessary to construct models that reduce this complexity to such a level that it enables us to understand the essential pro-
cesses that result in certain behaviors without oversimplifying reality. As I have argued throughout this dissertation, current models of personality and of psychopathology are oversimplifying reality: while convenient in that such models are relatively easy to understand, they defy reality by designating traits and disorders, for the existence of which we have no evidence whatsoever, to be the ultimate glue that holds together behaviors such as liking people and liking parties; or symptoms such as hypersonnia and fatigue. If we really want to know why people with certain personality features struggle more in daily life and why some of these people develop psychopathology as a result, I do not think that current (trait) theories will prove crucial in finding the answers. A network perspective, with its emphasis on direct interactions between thoughts, feelings and behaviors that result in certain equilibria, might be helpful; especially when, for personality, we further develop our intuitions about what thoughts, feelings and behaviors are suitable components for personality networks.

Whichever theory of mental disorders one adheres to, they all share a deep desire to understand the inner workings of mental disorders. We all agree that finding out why some people are more vulnerable to developing mental disorders than others, how we can protect vulnerable people from harm, and how we can effectively treat people who have already fallen into the abyss of mental dysfunction are among the most pressing questions in the fields of clinical psychology and psychiatry. A disease model of mental disorders likely will not bring us any closer to finding answers to these questions. The network perspective, for some disorders, very well might.